

Editorial

Cardiac hypertrophy in athletes

D OAKLEY

From the Department of Cardiology, Northern General Hospital, Sheffield

The outstanding physical ability of top athletes and the frequency with which world records are broken continue to fascinate layman and physiologist alike. It is generally assumed that such great achievements are the outcome of ever more rigorous and prolonged training programmes, which cause, among other things, adaptation of the heart in various ways. The most prominent of these is left ventricular hypertrophy. It is appropriate that in this Olympic year the significance of this hypertrophy should be discussed.

Different sports require different types of muscular effort. Endurance sports, such as running, involve mainly dynamic or isotonic effort. These sportsmen seldom appear as muscular as weight lifters, wrestlers, and throwers, in whom resistive or isometric exercise is important. Cyclists perform dynamic exercise with the legs and resistive exercise with the arms. In disease states there are different responses of the left ventricle to pressure and volume overload, and, by analogy with these, workers have sought differences in the cardiac hypertrophy between resistive and endurance athletes. The results have not been consistent. Some claim to have shown that endurance athletes undergo cardiac dilatation and hypertrophy, thus preserving the ratio of left ventricular wall thickness to cavity radius within the normal range. In "resistive" athletes the wall thickness was disproportionately large.¹ One group go so far as to liken the first group to congestive cardiomyopathy and the latter to hypertrophic cardiomyopathy (although the implication that the hypertrophy could be pathological is not justified).² Other workers have failed to show these differences.

As with many controversial subjects, there are now numerous published reports, and two more papers contributing to the debate are published in this issue of the *British Heart Journal*. At first sight their

findings would appear to be contradictory. Shapiro³ shows no significant differences between strength and endurance athletes, whereas Fagard and coworkers⁴ do. Both authors have interesting and novel approaches to this subject, although echocardiography remains the investigation of choice. Shapiro studied a large group of athletes and corrected his variables for body surface area. He found that these variables were not distributed bimodally as might be expected if two types of hypertrophy existed. He included a wide range of different sports and graded subjects according to their sporting status. There was considerable variability in this. Small groups of sedentary people and ex-sportsmen were included as control subjects.

Fagard and coworkers, on the other hand, studied two uniform groups. The choice of cyclists was important since their chosen sport involves many hours of isometric exercise for the arms. Considerable effort was made to ensure that the exercise capacity of the two groups in terms of oxygen uptake was comparable. This is a significant point overlooked by some previous authors. Two well chosen control groups were included. Body weight rather than surface area was used as a correction factor, but this would tend to be more rigorous in excluding differences attributable to variable build. The authors only speculate that the varying degrees of isometric exercise account for the differences observed and rightly point out that it would be necessary to estimate blood pressure during exercise to establish the mechanisms involved. Invasive blood pressure monitoring in these subjects is not easily justified.

The degree of cardiac hypertrophy in response to exercise differs between individuals. This may, in part, be due to inherited factors and also to the degree of physical training, which is difficult to quantify. There is overlap between the training programmes of different athletes; weight lifters run, runners lift weights. These problems account for many of the dis-

Requests for reprints to Dr D Oakley, Department of Cardiology, Northern General Hospital, Sheffield S5 7AU.

crepancies in published reports, but careful studies such as that by Fagard and coworkers, in which as many variables as possible are standardised, suggest that differences between various types of athletes do exist.

The time now seems right for a change of emphasis in the study of cardiac hypertrophy in athletes. Descriptive studies abound, but the significance of the changes described is not understood. Shapiro concludes that hypertrophy in athletes should not be considered unphysiological. Future lines of research should aim to confirm or refute this conclusion. What is the time course of the development of hypertrophy in response to training? How quickly does it resolve or regress when training stops? Does this hypertrophy, and particularly the more extreme examples, carry an adverse prognosis? Is there an association with sudden death?

Longitudinal studies on athletes are sparse. Shapiro implies the importance of this aspect by including ex-athletes in his report, but unfortunately their previous cardiac dimensions are not known. Fagard and coworkers have reported seasonal variations in the cardiac dimensions of cyclists corresponding to the resting and active seasons.⁵ Other workers have shown quite rapid progress in cardiac dilatation and hypertrophy in response to training, which is particularly prominent when an unfit individual starts training; this has been noted, for instance, in oarsmen.⁶ Nishimura *et al* studied Japanese cyclists who were still actively training. The older subjects had more hypertrophy than their younger compatriots, and there was some slight depression of left ventricular function, which was of uncertain significance.⁷ The conclusion from these studies is that hypertrophy can develop and regress quite quickly—that is, over a few months. Regression during the resting season is incomplete, and older subjects may develop more hypertrophy for uncertain reasons.

There is now a need for a large scale prospective study of cardiac hypertrophy in athletes with a very long follow up period. Hypertrophy will need to be documented by electrocardiography and echocardiography and regular follow up maintained after the athletes stop training and competition. Particular attention should be paid to those with more extreme hypertrophy to establish the reversibility of this and any adverse outcome. Such a study would not yield immediate results and should be carried out with the full support of those institutions concerned with the welfare of athletes.

In the absence of such a study, as outlined above, can anything be said about the prognosis of athletes, and does an increased wall thickness to cavity radius ratio affect this adversely?

As a group, athletes are healthy and long lived.⁸

The unexpected death of an athlete is a catastrophe which attracts notice and comment. The incidence of sudden death in athletes, however, is not known so it cannot be compared with that of an age matched sedentary population. The group in Bethesda have looked at the problem of sudden death in athletes, but naturally they were forced to do so retrospectively.⁹ They emphasise the frequency with which cardiac lesions were found at postmortem examination. Changes resembling hypertrophic cardiomyopathy were seen, but relevant clinical information about the subjects was incomplete. It is not clear, therefore, whether training had induced a situation resembling hypertrophic cardiomyopathy in these people or whether they had this condition from the start and their athletic prowess was incidental. The former possibility would be more sinister. A distinction could perhaps be made in living subjects by studying the reversibility of asymmetrical hypertrophy on cessation of training and also by documenting cardiac dimensions in first degree relatives. Isolated reports suggest a considerable potential for regression,¹⁰ but a more systematic study is required.

Even if the victim of sudden death appears to have a normal heart, other conditions such as the Wolff-Parkinson-White or long QT interval syndromes (unrelated to athletic training) could have been responsible. Only prior medical examination might have detected these abnormalities.

Some athletes tend to be introspective and worry about their health. They present to doctors with minor complaints such as musculoskeletal chest pain, or an odd electrocardiogram is found at a routine examination. Equivocal opinions and advice fail to allay these anxieties. There is at present no published evidence to suggest that training induced hypertrophy of whatever type represents anything other than cardiovascular fitness. The greatest degrees of hypertrophy may require further investigation. If a positive diagnosis of cardiomyopathy cannot be made the patient must be vigorously reassured that he has no abnormality.

References

- 1 Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. *Ann Intern Med* 1975; 82: 521-4.
- 2 Sugishita Y, Koseki S, Matsuda M, Yamaguchi T, Ito I. Myocardial mechanics of athletic hearts in comparison with diseased hearts. *Am Heart J* 1983; 105: 273-80.
- 3 Shapiro LM. Physiological left ventricular hypertrophy. *Br Heart J* 1984; 52: 130-5.
- 4 Fagard R, Aubert A, Staessen J, Eynde EV, Vanhees L, Amery A. Comparative echocardiographic study of car-

- diac structure and function in cyclists and runner. *Br Heart J* 1984; 52: 124-9.
- 5 Fagard R, Aubert A, Lysens R, Staessen J, Vanhees L, Amery A. Noninvasive assessment of seasonal variations in cardiac structure and function in cyclists. *Circulation* 1983; 67: 896-901.
 - 6 Wieling W, Borghols EAM, Hollander AP, Danner SA, Dunning AJ. Echocardiographic dimensions and maximal oxygen uptake in oarsmen during training. *Br Heart J* 1981; 46: 190-5.
 - 7 Nishimura T, Yamada Y, Kawai C. Echocardiographic evaluation of long-term effects of exercise on left ventricular hypertrophy and function in professional bicyclists. *Circulation* 1980; 61: 832-40.
 - 8 Pomeroy WC, White PD. Coronary heart disease in former football players. *JAMA* 1958; 167: 711-4.
 - 9 Maron BT, Roberts WC, McAllister HA, Rosing DR, Epstein SE. Sudden death in young athletes. *Circulation* 1980; 62: 218-29.
 - 10 Oakley DG, Oakley CM. Significance of abnormal electrocardiograms in highly trained athletes. *Am J Cardiol* 1982; 50: 985-9.